THE REACTIONS OF INJURED HUMAN ARTICULAR CARTILAGE

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The physical and chemical structure of normal hyaline articular cartilage was described by Collins (1949), but there is little published on its reactions to injury and repair in the human subject, though animal observations have been made, notably by Bennett, Bauer and Maddock (1932). The slow metabolism and physiological inactivity of joint cartilage were emphasised by Bywaters (1937), again mainly on animal material, and the general view is that if cartilage has any reactions at all they are so slow that they can be neglected. For two reasons these reactions seem to call for investigation: firstly that in the prolonged course of human arthritis even a slow reaction cannot be neglected; and secondly because the study of the reactions of a substance of relatively known structure under physiological conditions may throw some light on the methods by which the changes of this substance in the arthritic diseases are brought about.

In this paper the changes that follow simple mechanical injuries to the joint cartilage are considered. The affected surfaces were removed at operation or necropsy after intervals from three days to ten years after the original injury; the ages of the subjects lay between twelve and fifty-nine years. The material available included sixteen fractures of the head of the radius, eleven of the patella and smaller numbers of other fractures.

NORMAL STRUCTURE

The terms used by Collins (loc. cit.) for the zones of cartilage are self-explanatory. For the wavy line that marks the junction between calcified and uncalcified cartilage—a feature to which reference will frequently have to be made—the term "tidemark," introduced by Fawns and Landells (1953), is conveniently brief.

It is important to keep in the foreground the fact that the articular end of a bone is not a separately made hinge-piece attached to the rest, but is built up in the adolescent epiphysis by growth and ossification over a period of twenty years before it reaches a mature state. In the young child there is no continuous subchondral osseous plate under the thick cartilage, but loci of ossification which come to lie closer together until, in adolescence, a continuous lamella is found; this thickens in adult life in accordance with the stresses and pressures upon it. Only then does a definite zone of calcified cartilage appear beyond the bone. In this zone blood vessels are unusual, though they were described by Holmdahl and Ingelmark in rabbits (1950), and by Harrison, Schajowicz and Trueta in man (1953); they are too few to have much responsibility for cartilage nutrition and are separated from the cartilage by a thin bone lamella. These represent the last traces of active ossification that has been going on since childhood; this process, though dormant, is not effete and can be seen to become reactivated.

Along with the rearrangement of cells and the appearance of subchondral bone that takes place with increasing age, there are conspicuous changes in the cartilage matrix. In the foetus and young child there is little collagen but much polysaccharide; this is in the phase characterised by strong metachromasia with Toluidine blue but a very weak or absent Periodic acid–Schiff reaction. As age advances, the PA–S reaction becomes stronger, the metachromasia less intense and complete, though never lost in health. The collagen component becomes increasingly obvious, staining deeply with van Gieson’s stain; but where the metachromasia is deepest and most intense there is less collagen stainable with van Gieson’s stain.

548 THE JOURNAL OF BONE AND JOINT SURGERY
It is probably legitimate to suggest that in growth the cartilage cells first form an excessive amount of highly polymerised and sulphated acid mucopolysaccharide. The first fine fibrils of collagen added to this—well below the limit of visibility and hard to demonstrate with the electron microscope—will have their relatively few basic groups saturated with the excess of acid mucopolysaccharide, and so will not show the usual red staining with eosin or with van Gieson; but if the proportion of chondrin to collagen is greatly decreased, whether because too much collagen or too little chondrin is being made, the collagen will then become acidophil. It seems further that at some stage part of the chondrin loses its metachromasia and part—possibly, but not necessarily, the same part—becomes PA--S positive. These changes are commonly seen in older cartilage, both with and without osteoarthritic changes.

The considerable variations in the shape of the cartilage cell, and the extent and shape of the metachromatic patch around it in apparently healthy cartilage, are evidence of the variable physiological state of this cell which is by no means inert when time is granted to its slow processes.

![Cartilage Image](image)

**FIG. 1**
Pott’s fracture, three weeks old. Woman aged twenty-eight. Tibial articular surface. (Periodic acid-Schiff, × 28.) Separation of uncalcified cartilage along the “tiremark” ; fibrinous debris in the cleft; bone and calcified cartilage below with callus in the marrow spaces. Fracture completely through bone to the right. Tear in section to the left made during preparation; this remains empty.

**OBSERVATIONS ON COMPLETE FRACTURES**

**Immediate changes:**

1. The lines along which the cartilage breaks are those of the fibres described by Benninghoff (1925). Histological features are so often dismissed as artefacts that this independent confirmation of their reality is valuable.

2. In addition to these lines of weakness there is an important additional one lying along the plane of the tidemark (Fig. 1) which is commonly found and has not been commented on. This is the level at which almost all transverse fractures are found. Elsewhere they tend to run vertically through cartilage, calcified cartilage and bone. In particular, there is no tendency to split along the more obvious line of the bone-cartilage junction, where weakness might be expected. This peculiar weakness of the tidemark in one direction is accompanied by increased strength at right angles to it: thus splits from above and erosions from below have a natural
tendency to stop at the tidemark, and if frozen sections are handled this zone may hang together like a cord when the rest of the section is torn. It must be emphasised again that this is not a structurally separately built membrane, but develops in cartilage during adult life as calcification occurs deep to it. The striking difference in physical qualities associated with the adjacent calcification suggests that considerable reorientation of fibres and bonds occurs with calcification.

3. The physical characteristics of plasticity and coherence in uncalcified cartilage are shown well in some fractures. In the specimen shown in Figure 2 the folded piece of cartilage has been torn off at the tidemark, rotated and bent double, so that the original articular surface has been turned towards the interior of the bone. In spite of this violent displacement there is almost no fragmentation.

4. The zone of dead cells around the site of injury is exceedingly narrow; virtually only

![Figure 2](image_url)

Fractured head of radius nineteen days old. Woman aged fifty-six. (Haematoxylin and eosin, x 16.) Extreme displacement, buckling and rotation of cartilage without fragmentation. Early invasion and digestion by granulation tissue below on the left and at the right-hand edge. The dark line of the tidemark can just be made out in the middle.

the cells directly in the line are killed. The elasticity of cartilage is such that brief violent compression is not necessarily fatal.

**Intermediate changes**—The following observations were made from sections examined three to fourteen days after the injury.

1. There is no tendency for the cartilage to flake around the margin of the injury. This is important in relation to the pathogenesis of osteoarthritis, which is sometimes attributed to disruption of the surface layer, allowing the lower layers to separate. Observation shows, however, that mere mechanical severance of surface layers in healthy cartilage is not of itself sufficient to make the whole incoherent. It will be different if the cartilage is already unhealthy. Longer-term observations where the joint has been subjected to the strain of normal use confirm this: thus, in a shoulder examined seven years after a Nicola’s operation for recurrent dislocation, there was no roughening of the cartilage round the opening made in it for the long head of the biceps.

2. There is very little multiplication of the cartilage cells exposed by the breach. Since it is
generally conceded that their nutrition is mainly synovial, it might be expected that the improvement in nutrition brought about by direct access to the synovial fluid would stimulate their multiplication, but even in young humans little of this occurs, though I have seen it to a limited degree in young animals. Here again the import is related to osteoarthritis: the multiplication of cells observed in fibrillated cartilage cannot be explained simply by their improved access to synovial fluid.

**Late stage of repair**—The following changes were observed in specimens examined fourteen days to ten years after the injury.

Two events require consideration: 1) the replacement of gaps left in the articular cartilage; and 2) the fate of displaced fragments of articular cartilage.

1. Replacement is invariably by fibrous tissue, derived from granulation tissue emerging from the depths of the bone if the gap is central, from the synovial membrane if the gap is peripheral, or both. This fibrous tissue, if exposed to the synovial fluid, is eventually impregnated with polysaccharide and may in years come to resemble fibrocartilage and present a remarkably smooth surface, but the internal structure is not that of hyaline cartilage formed since infancy. Repair by genuine hyaline cartilage has not been seen in the present material.

The original hyaline cartilage at the edge of the breach contributes nothing to the new tissue; the line of demarcation is as clear ten years later as it is at the time of the injury (Fig. 3).

When for any reason the synovial fluid cannot reach the fibrous tissue, the resemblance of the fibrous tissue to cartilage is less close—for example the modified fibrous tissue that fills up the space between the cup and the neck of the femur in the operation of cup arthroplasty.

The fibrous cartilage formed in repair is not inefficient, though it has not the structure of the original hyaline. The interlacing coarse-fibre bundles render it less liable to the splitting which disrupts the fine parallel fibres of the usual cartilage in osteoarthritis. Fusion between the new and the old may be intimate, with the immigration of fibrocytes into cartilage matrix, or they may merely lie closely together, with a tendency to split on handling, as in Figure 3.
Bone or calcified cartilage exposed in the joint without fibrous tissue overlying it does not appear to survive. The bone dies when nourished only by synovial fluid, and both bone and cartilage become mechanically eroded and abrade the opposing face. They also form a barrier that granulation tissue is slow to remove, osteoclastic resorption being necessary, and this is seen also in purulent digestion of cartilage. The clinical sequel of osteoarthritis after joint fractures is more attributable to this, to malalignment of the bones, or to changes in the hyaline cartilage of the rest of the joint, than to the fibrocartilage of repair.

2. Displaced cartilage is treated in several ways. When fibrous tissue grows over the surface and is itself nourished by synovial fluid, the new stratum of fibrocartilage may bury the old, which survives almost unchanged beneath it, as seen in Figure 3 seven years after the injury. This is strong evidence of the low energy requirements of hyaline cartilage, here satisfied by diffusion from the joint space through double the usual thickness of material. When, however, the cartilage is displaced into the granulation tissue of the callus, it does not survive. In the first three weeks little appears to be taking place round it, compared with the activity around fragments of bone; by six weeks the cells are dying, the chondrin leaches away and the fibrous tissue stains increasingly feebly; the cartilage still remains coherent and hyaline, and may be found in that state many years later, with bone deposited over it.

More usually, however, cartilage perishes wherever it is in contact with granulation tissue. The cells die and the chondrin is dissolved away before actual invasion has gone far. The chondrin may be found absorbed on to neighbouring collagen strands; these are metachromatic only when they are close to cartilage that has recently lost its chondrin. Later, immigrant fibrocytes penetrate the matrix and replace it by fibrous tissue, which may ossify. These cells are capable of invading living cartilage, in which case the cells of the cartilage lose their identity but do not always die. More often the cartilage is dead already when it is invaded, the cells undergoing lysis though their spaces may remain long visible. This death appears related to the near approach of the granulation tissue, as the zone of death is distributed around the edge of the cartilage and is greatest near the granulation tissue—exactly the reverse of what would be expected if anoxia or starvation were concerned.
The reactions of injured human articular cartilage

(Figs. 2 and 4). This is in keeping with the known low oxygen requirements of cartilage. No foreign cells are required to effect this removal. In calcified cartilage, erosion is associated with the presence of cells like osteoclasts, and no solution of the chondrin takes place (Fig. 5).

It is not possible to associate the death with any particular component of granulation tissue. Polymorphonuclear leucocytes are not concerned in it. The fate of small fragments of cartilage which are dislodged into the joint was described by Lloyd-Roberts (1953); they are swept by movement into the angles of the joint and engulfed by synovial cells.

The evidence from these "experimental human autografts" is the more valuable because serological complications and operative sepsis can be excluded. The violence of the injury remains uncontrolled and, since this will be greatest on the surface of the fragments, it is possible that the death of the cartilage cells may in part precede the arrival of the granulation tissue and may indeed induce the latter. The fact that dead fragments are not seen before the granulation tissue is not incontrovertible evidence that granulation tissue is fatal to cartilage, since chemical changes may precede the visible ones. But the narrowness of the zone of death seen immediately after the injury, and similar changes seen when gross trauma can be excluded (vide infra) are stronger evidence.

Bone is often deposited directly on the surface of the displaced cartilage, either alive or dead, calcified or uncalkified. Calcified cartilage, lying deeper, is the more common substrate, but it is of theoretical importance that calcification is not essential. Fine bone slivers can often be seen passing beyond the tidemark. It is often stated that provisional calcification is essential as a preliminary to ossification, and calcification is thought to be indicative of impending ossification. These statements are not always true, and the processes should be

FIG. 5
Fracture of head of femur two months old. Girl aged sixteen. (Toluidine blue, x 38.) Necrosis of cartilage and bone. Complete removal of chondrin from uncalcified cartilage; no change in the calcified zone.

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considered as independent though often associated. Calcification is essential for the piecemeal removal of lumps of cartilage by osteoclasts, as distinct from the infiltrating type of destruction by granulation tissue described above. Part of the trouble in the rachitic epiphysis arises from this difference in the removal of the two types of cartilage.

When bone is formed over dead uncalcified cartilage, a haze of calcium granules may be observed to develop in the cartilage. Since there are no visible living cells in the cartilage, this cannot be an active step of deliberate provisional calcification by cartilage cells but must represent a diffusion of calcium from the forming bone. It does not occur in cartilage that has no bone on the surface, and thus cannot be due to absorption of calcium from the blood. It has been suggested elsewhere (Fawns and Landells 1953) that this is the method of formation of the normal calcified zone that lies between the adult articular cartilage and the bony articular lamella.

![Fracture of osteoarthritic patella](image)

**FIG. 6**

Fracture of osteoarthritic patella a few days old. Woman aged fifty-three. (van Gieson, x 28.) The fracture is just out of the picture to the right. A cleft containing fibrin-like material crosses nearly the whole section at the tidemark.

**OBSERVATIONS FROM INCOMPLETE FRACTURES**

When the stress on the joint is insufficient to cause clinical fracture, damage may be found on microscopy, necessarily in areas chosen at random or for other investigations; proof that they are indeed traumatic will be inferential, and it will not be possible to refer the injury to a definite date. Articular cartilage, consisting of an elastic surface layer over a thin calcified brittle one, would be likely to slip at the plane between these layers, and the existence of a plane of weakness at this level has been commented on. Moreover, if there is a gap in the deeper tissue the elastic cartilage might be expected to herniate into the opening. Such changes can be seen in sections, without any other structural abnormality, sometimes near complete fractures but also independent of them, and it is reasonable to suggest that they are mechanical. Three stages, short of complete fracture, can be described: 1) splitting at the tidemark; 2) depression of cartilage into the bone; and 3) fissuring of both calcified cartilage and bone along a vertical plane which permits blood vessels from the marrow to reach the cartilage.
Tidemark splitting (Fig. 6)—These fissures occurring during life are marked by the accumulation of a pool of acellular material; accidental fissures occurring in the preparation of the section are empty. This material has some of the standing features of fibrin: it is strongly PA–S positive, PTAH positive, picrophil in van Gieson's stain; it is not metachromatic, does not dissolve in collagenase, or lose its staining reactions after hyalase.

Impression of cartilage into a vertical fissure (Figs. 7 to 9)—There is usually associated deformity in the adjacent bone and cartilage. The displaced cartilage is unusually deeply stained with basic dyes, unusually resistant to enzyme digestion, and often mixed with the eosinophil material described in the previous paragraph. The cells may either proliferate or atrophy but rarely remain unchanged.

Vascularisation through fissures—Though this is believed to be traumatic in many cases, once the process is established it will closely resemble the picture resulting from reactivation of ossification where it had stopped after adolescence in the articular lamella. In this process the bone that has been laid down will first be breached by osteoclastic resorption, but thereafter traumatic and erosive breaches will look alike, and for that reason the neutral term "intrusion" of blood vessels has been adopted. The consequences of the two events leading to intrusion are similar.

An early but clear example is shown in Figures 10 to 12. The points of particular importance are: 1) Capillaries grow directly into the uncalcified cartilage without the mediation of any other cell to destroy the matrix ahead of them; 2) there is physical splitting of the calcified cartilage behind them, but no invasion or solution of it; 3) the shape of the intruding mass into the cartilage is smoothly rounded; 4) there is well-marked zonation around this—a ring free of chondrin next to the untouched cartilage; next a ring where there is a gathering together of fine fibres to form a coarser set of strands—these are picrophil and strongly metachromatic; and finally among the capillaries the fibres become very large and irregular and the cartilage cells disappear. This zonation is not conspicuous in the PA–S stain. This process and the events taking place around displaced cartilage are similar.

The smooth rounding of the unit suggests that the capillaries act as a whole, not as individual foci of erosion, and also that diffusion is one factor in the process. The simple hypothesis is that there are two fractions of the carbohydrate, a metachromatic mobile one and a fixed PA–S positive one; and that the first result of vascular intrusion is the elution of the chondrin faster than the cells make it, giving the pale ring and permitting the collagen fibres to aggregate into the coarser strands visible in the section. On these coarser strands some at least of the chondrin becomes temporarily readsorbed, so giving the metachromatic-picrophil zone, and preventing the acid dyes from access to the collagen. The PA–S component remains uniformly visible throughout. Under these vascular conditions the cartilage cells either lose their identity or die, the conditions both as regards diffusion currents and oxygenation differing greatly from those in the depths of avascular hyaline cartilage to which they are accustomed.

A rare variation of this picture is shown in Figure 13. There is a dark basophil, but not metachromatic, ring between the pale ring and the zone of twisted "spun" metachromatic strands. This again is related to the focus as a whole and not to any particular component of it; it has the uniformity and smooth outline of a soluble rather than of a particulate material. The intensity of the stain suggests a strongly acidic substance, possibly a modification of the chondrin diffusing one way and meeting other substances coming up from the blood. It is not calcium, which has a granular texture. Like the other changes, it stops abruptly at the edge of the calcified cartilage.

When the process has gone on further, the degeneration of the hyaline cartilage is more complete. In Figure 14 an arteriole is present at the breach through the calcified cartilage. The hyaline cartilage has lost its chondrin and reverted to fibrous tissue, which has broken up into a tuft in the middle of the patellar cartilage very like the fibrillation of osteoarthritis.
Fractured head of radius six weeks old. Man aged twenty-seven. (Toluidine blue, × 29.) Gross displacement near line of fracture. Disruption of calcified cartilage and displacement of both calcified and uncalcified cartilage into the bone, the latter proliferating (the tongue of medium grey tissue in the centre).

Fracture of patella seventeen days old. Man aged twenty-eight. (Periodic acid-Schiff, × 120.) About 1 centimetre from the fracture line; patella apparently healthy. Two gaps in the (slightly paler) calcified cartilage, in both of which uncalcified cartilage is present. Proliferation of cells in the left displacement and ossification in both. Traumatic origin of displacements is likely.
The chondrocytes, as far as they are recognisable in the margins of the intrusion, have reverted to fibrocytes, and there is much more than normal collagen in the area; this process represents not only removal of one component of cartilage but includes excess production of the other.

The usual sequel is the early sealing off of the intrusion by ossification. This results in the formation of a nodule of bone in the deeper cartilage which, by fusion of adjacent foci, advances the bony articular lamella and thins the overlying cartilage in the middle of the joint: this will impair its elasticity and favour further injury. At the edge of the cartilage it results in osteophyte formation.

DISCUSSION
Relation between articular cartilage and blood supply—It was pointed out first by Strangeways (1920) and Fisher (1922), and is indeed a commonplace observation in joint pathology, that cartilage may live, grow and calcify (but not ossify) when completely detached from the bone, provided it has free access to the synovial fluid. Because of this and the absence of blood vessels in adult articular cartilage, it is generally accepted that the normal nutrition of cartilage is mainly synovial, though there is evidence of some diffusion from the bone. The presence of occasional blood vessels in the subarticular plane has been interpreted as of importance in the normal nutrition of cartilage, but they are few in number and are insulated by a thin sheet of bone.

If the blood vessels are present without this sheet, abnormalities are visible in the cartilage. Examples of such contact have been given arising in each of three ways—trauma displacing fragments into callus, vascular tissue growing over the surface, and intrusion of blood vessels through the articular lamella from trauma or erosion. In each case the cartilage is being destroyed. It would appear that so far from being necessary for the life of the cartilage, a good blood supply is rapidly followed by complete disorganisation of hyaline cartilage.
and its replacement by fibrous tissue, fibrocartilage or bone. The exiguous nutrition by
synovial fluid to which cartilage is adapted is apparently a condition of its health, and the
richer standard of living provided by the blood stream is too generous for it. Some
confirmation of this view was given by Subba Rao (1954) who observed that cartilage grafts
from the xiphi sternum of rats were absorbed by granulation tissue within twelve weeks, if
they were small. Larger grafts developed a dense perichondrium and persisted; one became
ossified.

**Mechanism of cartilage removal**—No specialised phagocyte appears in the removal of
uncalcified cartilage, although when collagen is being removed, for example in suture material
or tendon, giant cells are usual. It is possible that the collagen is in fact not removed, but
that the solution of the surrounding chondrin changes the physical state of the collagen, so
that fine fibrils dispersed in a gel come to form tight bundles (Figs. 10 to 13). These take up
little room compared with their immediately adjacent pale ring, possibly explained by the

![Image](https://via.placeholder.com/150)

**Fig. 10**

Patella excised for chondromalacia. Woman aged thirty. (Periodic acid-Schiff, x 530.) The capillaries are close up against the face of the cartilage
undergoing solution: five cartilage cells present amongst them but no
adventitious phagocytic cells. Expansion of the blood vessels into the
uncalcified cartilage above, but limited below by the tidemark.

loss of much bound water as well as the acquisition of fresh interfibrillar bonds. In this
vascular digestion the adjacent calcified cartilage undergoes no change; its removal is always
associated with the presence of multinucleate giant cells. It is therefore likely that calcium
stabilises the linkage between collagen and chondrin in the natural removal of cartilage just
as it does in enzyme digestion in experimental conditions (Fawns and Landells 1953).

The relation between collagen staining red with van Gieson's stain and with eosin on
the one hand, and collagen without metachromasia and therefore probably free of chondrin
on the other hand, suggests that the chemical basis of the attachment of the acid dyes and
the acid mucopolysaccharides is the same, and is available for one or other purpose alternatively,
but not for both simultaneously. This alternation is seen in arthritic cartilage as well as in
the events discussed in this paper. The form and distribution of the PA-S staining material
and its digestibility are those of the collagen, and it is therefore suggested that this material,
probably polysaccharide, is intimately linked to the collagen.
Relation to osteoarthritis—The frequency with which the changes described as "incomplete fractures" are found is much higher in material that shows naked-eye evidence of osteoarthritis than in joints with a perfectly smooth surface. Trivial defects are common enough in controls; severe ones are nearly always found in osteoarthritis, but are not confined to the badly affected parts of the joint surfaces. They can be more easily interpreted as mechanical processes than as either degenerative or reparative. They are clearly associated
Another intrusion from the same bone. (Toluidine blue, ×440.) Invasion of cartilage by capillaries from the bone below, with pale diffusion zone limited by a dark basophil ring. Cartilage cells and the aggregation of collagen fibres well seen in the next zone; no adventitious cells. Process mechanically checked in the calcified zone but expands in the soft uncalcified zone; chemical changes stop abruptly at the tidemark.

Patella from a man dead of disseminated lupus erythematous, aged thirty-four; apparent chondromalacia; no known injury. (van Gieson, ×12.) Gap in the centre through which an arteriole passes. Elution of chondrin; disorganisation of hyaline cartilage and reversion to fibrous tissue in a V-shaped zone centred on the breach. Lines of cartilage-bone junctions on both sides are level; pattern of bone symmetrical; gross fracture is unlikely to have been the cause. Joint elsewhere normal.
with osteoarthritis, but whether as cause or effect is less certain. Minor traumatic events might well have a more serious effect on cartilage that was already degenerate, and inelastic or eroded cartilage will expose the underlying bony lamella to unusual strains which will permit vascular intrusions. The two processes are mingled, but there is some evidence that trauma alone will account for degenerative changes in healthy cartilage, and that frequently repeated minor injuries in ordinary life can so affect the vascularity of cartilage that the changes of osteoarthritis will follow.

Repair of the damaged cartilage of osteoarthritis would require, first, removal of the old material, and second, the free access of vascular tissue to the lacuna. In the body this is denied first by the dense bony lamella which has undergone thickening during the degenerative phase; and if this is penetrated (which is relatively uncommon) the granulation tissue forming will be crushed out of existence by the pressure from the opposite joint surface. Indeed it never achieves more than a local nodule of fibrocartilage and does not flow over the whole bare area. A conspicuous exception to this is the thick fibrocartilaginous cap which forms under the protection of the Vitallium cup in the Smith-Petersen arthroplasty. Here the preparation of the upper end of the femoral neck opens the vascular part of the bone widely and spreads any pressure uniformly. The result is a thick cap of coarse-fibred bluish white material which makes a very effective replacement of the hyaline cartilage, especially when after a few years it is saturated with mucopolysaccharide. The surgical intervention brings to completion the naturally unsuccessful efforts at cure. In the elderly, the vigour of growth is reduced and the available time for the slow repair of cartilage is short; even in young subjects repair is much better seen in the parts of the joint nearest the synovium. This fibrocartilage of repair can be distinguished both on its internal structure and on the appearances in the underlying bone from the degenerative fibrillary changes in osteoarthritis, though it is sometimes confused with it.

SUMMARY

1. The lines of fracture confirm the suggestions of earlier authors on the lines of strength in cartilage, with the additional feature of a transverse plane of weakness at the apex of the calcified zone.
2. The normal nutrition of cartilage is synovial, and access of a free blood supply is followed by destruction of hyaline articular cartilage.
3. Minor traumatic events in the articular lamella are common, particularly in osteoarthritic joints; the results of these on the cartilage are like the changes of osteoarthritis.
4. The removal of uncalcified cartilage can be described in two stages of a physico-chemical kind; the removal of calcified cartilage is a single cellular process.
5. There is evidence that the carbohydrate moiety of cartilage is present in two separable phases, one fixed to collagen, the other free.
6. The repair mechanisms after fracture are those available to restore the damage of osteoarthritis, and reasons can be shown why in fact they are ineffective.

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REFERENCES


